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Review

Physiology and pathology of TASER® electronic control devices

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ABSTRACT

TASER® ECDs (electronic control device) are small, battery powered, handheld devices. They deliver short duration, low energy pulses to stimulate motor neurons, causing transient paralysis. While the experience is painful, proper use of the device is rarely associated with significant side effects in spite of 1070 human worldwide exposures daily. In fact, there have been more than 780,000 training exposures and 630,000 field uses (total of over 1.4 million human uses) without any credible evidence of a resulting cardiac arrhythmia. In this article we describe the mechanisms by which the device operates, and review possible morbidities.

1. Introduction

1.1. Relevant electrical principles

Electrical current is best conceived of as the number of electrons going down a wire per second. Since the number is typically very large, the term "ampere" (abbreviated "A") is used to represent it and this represents 0.24×10^{18} electrons per second. The term "voltage" merely refers to the electrical pressure pushing the current down its path. One volt (V) is roughly the voltage from a single battery cell. Pathways with high resistance allow less electrical current for the same electrical pressure or voltage. The resistance unit ohm is simply given by the voltage divided by the current.

Continuous currents flowing through the human chest of 0.3 A are typically fatal. Yet a strong static electricity shock can have 100 times the peak current – 30 A – and produce no residual damage.[1] The reason that the 30 A peak static shock is not fatal is that the pulse of the static electrical discharge is too short in duration to affect the heart muscle or cause an arrhythmia.[2]

The term "electrical charge" refers to the area under the electrical current curve. It represents the total number of electrons delivered in a pulse. A charge of 1 C is defined as 6.24×10^{18} electrons. The charge from a TASER X26 ECD (Electronic Control Device) pulse is approximately 100 μC (microcoulombs) or about 6×10^{14} electrons.[3]

1.2. The TASER ECD waveforms

The TASER X26 ECD is a pistol shaped device weighing 205 g. It has a limited power source (a battery of two lithium camera cells – Duracell® CR123 – of 3 V each), and shoots two tethered probes. Together they deliver 19 very short duration pulses per second (19 PPS), with a typical peak voltage of 1900 V (1400–2600 V), over a 5-S pulse voltage is actually only 600 V.[4] The device also generates an open-circuit voltage of up to 50,000 V to arc through air or across thick clothing but that voltage is never seen in, or "delivered into" the body.

It is certainly not intuitively obvious how a 50 kV pulse can pass through clothing but then not pass through the body. The technical reason is that the clothing has a very high impedance (nearly infinite) while the body has a much lower impedance (on the order of 600 ohms). In addition, the 50 kV pulse is generated by separate output circuitry from that of the 600 V pulse. The 600-V pulse is generated by a direct connection of charged capacitors which deliver a fixed charge to the body. The 50 kV pulse is generated by a high-impedance transformer output which has low current capacity. An analogy is the van-de-Graff generator which delivers a high voltage – but low current – arc. While impressive, it cannot power a simple lantern while a high current – but low voltage – 1.5 V battery can.

While incapable of stimulating muscles, the "impressive" nature of the arc and the natural fear of electricity adds a significant additional capability to the ECD. Police officers in the United Kingdom have found that the mere display of the arc is enough to gain compliance in the majority of cases.[5]

The pulse generated is specially designed with a very short duration of 100 μs (microseconds) to efficiently capture alpha motor neurons.[2]

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The TASER ECD should not be confused with generic "stun guns." Those devices deliver less average current (typical values of 0.3–0.5 mA) and deliver it over a short pathway between two fixed electrodes. The result is pain, but they lack both the electrical charge and the electrode spread to cause motor neuron activation and skeletal muscle capture. With stun gun electrodes only 2–5 cm apart – and the lack of skin penetration – the current flow is primarily through the dermis between the electrodes and there is no significant penetration beyond the fat layer. Thus there is insufficient current in the skeletal muscle layer to capture motor neurons and achieve muscle control.

The average current of the TASER X26 is approximately 1.9 mA (milliamperes) = 19 pulses per second $\cdot$ 100 μC. (This is about 5 times the average current of a typical stun gun.) As seen in Fig. 1, a typical peak current is ~2.5 A (range of 2–4 A).

The devices use compressed nitrogen to fire 2 small probes at typical distances of 7.7 m.[6,7] (Other TASER cartridge models can reach a distance of 11 m.) When the trigger is pulled, the high voltage first serves to open the nitrogen cartridges to release the nitrogen to propel the probes towards the target. These probes themselves are designed to pierce or become lodged in most light clothing (which generally offers no protection to the shock delivery due to the 50,000 V arcing capability). The sharp portion of the probe is typically 9 mm long and typically penetrates the epidermis and dermis by 4–5 mm for a good electrical connection.

The probe cartridge can be removed and the device used in a "drive-stun" mode by pushing the front of the weapon into the skin to function as a higher charge stun gun. The drive-stun mode is also available by retaining an expended cartridge on the weapon. Since there is insufficient electrode spread to capture muscles, the drive-stun mode serves only as a compliance technique.

1.3. How does the ECD cause temporary local paralysis?

Even as a strong static shock will temporarily incapacitate someone, a series of 19 very short duration shocks per second causes temporary incapacitation. The ultra-short electrical pulses applied by TASER ECDs are intended to stimulate Type A-α motor neurons, which are the nerves that control skeletal muscle contraction, but without stimulating cardiac muscle. There are many established reasons why the device should have no effect on the heart; these are enumerated in Table 1.

1.4. Which nerves are stimulated?

Current from ECDs is intended primarily to disable the target by preventing voluntary movement. The largest diameter myelinated

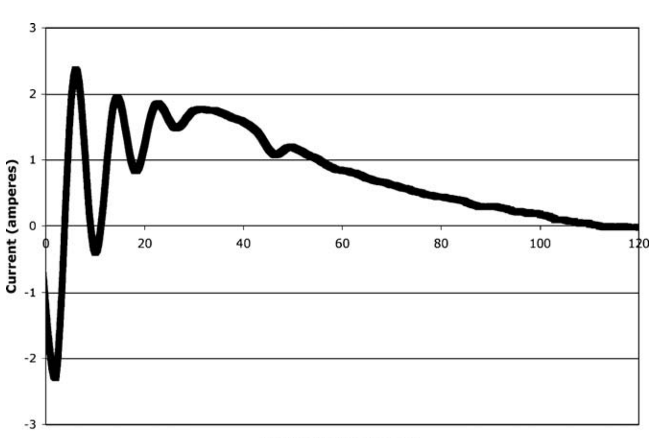

Fig. 1. Output waveform of TASER X26 ECD with probe deployment in human volunteer.

Table 1
Factors promoting cardiac safety in spite of skeletal muscle capture.

1. The anatomic location of the heart farther from the probes than the skeletal muscle nerves. Modeling studies show that the fields generated within the thorax are far too weak to have any effect on myocardial cells
2. Short durations of the electrical pulse compared to the chronaxie (optimal stimulus duration) of the heart
3. Anisotropy (tendency of current to follow the grain) of skeletal muscle largely wraps current around the thorax instead of into the thoracic cavity
4. Electrical shielding effect of the perpendicular muscle grain between the pectoralis major and minor, between pectoralis minor and intercostals, and between the intercostals and the epicardium
5. The electrical shielding effects of the lungs around large portions of the heart

A-α motor neuron axons (which innervate skeletal muscle fibers) tend to have relatively low electrical thresholds and are fairly easy to stimulate. This is because the stimulation threshold correlates inversely with cell diameter (so larger diameter cells are easiest to stimulate).[8,9]

Perceptions of discomfort, sensory overload, and pain are carried by myelinated axons (type III A-δ fibers responsible for the sensation of sharp pain) but also by small, nonmyelinated axons (type IV C responsible for dull, aching diffuse pain). The C fibers have stimulation thresholds about 20 times higher than those of sensory A fibers and thus the typical subject describes the effects as sensory overload and sharp pain. However, the primary physiological effect is the local paralysis while the discomfort is actually only a side effect.

1.5. Cardiac vs. muscle stimulation

By comparison to motor or sensory myelinated nerves, the electrical excitability of the heart is relatively low. This is because the cardiac strength-duration time constant (chronaxie) is about 3 ms (i.e. at least 10–20 times higher than the A-α motor neuron fibers which control skeletal muscle contraction).[10] This means that pulses much shorter than 3 ms must have much higher currents to stimulate the heart cells.

The heart is also located deep within the torso as opposed to the skeletal muscle which comprises much of the superficial layers. Fortuitously, electrical current prefers to follow the grain (fiber) of the muscles.[11] Thus the ECD current will tend to follow the grain of the skeletal muscle around – rather than into – the thoracic cage. Hence, relatively little, if any, current will pass through the heart.[12–14] This effect of relatively low penetration into the heart – given surface or near-surface stimulation of tissues – is well known and studied both in the electrical safety literature as well as the medical literature of transthoracic pacing and defibrillation.[15]

Both in terms of efficacy (efficiently activating motor neurons to affect skeletal muscle between and near the probes) and risk (producing current sufficient to cause ventricular fibrillation), the TASER X26 stimulus pulse appears to be ideal, since the former is maximal and latter minimal.

2. The ECD and the heart

2.1. Electrocution

The term "electrocution" was first coined to describe the judicial execution of a criminal by use of electricity – a contraction of "electrical execution". Today the term is more broadly used to describe the induction of ventricular fibrillation (VF) by the application of, or exposure to, electrical shock; death occurs virtually immediately. It is now generally accepted that an ECD cannot cause "electrocution" in an adult human.[16–21]

Primarily from the study of the ICD (implantable cardioverter defibrillator) – where induction of VF is a routine part of installa-

tion[22] – certain facts have been medically and scientifically established

a. VF is either induced or not induced within 1–5 s.[23–27]
b. There is no induction of the arrest-related death rhythms of asystole and PEA (pulseless electrical activity).[20,28–30]
c. The cardiac pulse disappears within seconds.[31]
d. The patient loses consciousness within 5–15 s.
e. A sufficiently strong defibrillation shock – either internal or external – restores a cardiac sinus rhythm 99.9% of the time.[27]

There is no increased risk of a later VF due to a preceding shock since electrical current is not stored in the body. This is in contrast to the common misperception that there is some increased risk from longer or multiple ECD exposures.

2.2. TASER ECD cardiac electrophysiology

There is no peer-reviewed data to support the notion that a TASER ECD could cause electrically induced cardiac death in a human. In any discussion of this subject, two factors must be distinguished: pulse duration and shock duration.

Pulse duration. The length of an individual electrical stimulation pulse. This is typically on the order of 1 ms or even far less. The pulse duration of an X26 ECD is about 0.1 ms or 100 μs.[2]

Shock or application duration. The total length of time that an electrical current is applied on the order of time that a human could estimate it. This is typically 1 s or more. For example, the programmed X26 application duration is 5 s. During that time it will deliver 95 = 19 PPS (pulses per second)·5 s.[2]

Contrary to popular belief, the 5-s programmed duration was implemented to give a minimum and not a maximum duration. This was due to early experience of police officers pulling the trigger once and releasing immediately as they would do with a firearm. Violent suspects were not disabled and injuries ensued. If the suspect is immediately controlled then the officer can flip the safety down and stop the application. On the other hand, if the suspect continues to resist, the trigger can be held down to give a longer application.

There are two ways to deliver an electrical shock that will induce VF. The first is to deliver a single shock that is sufficiently strong exactly during repolarization (the "T" wave).[32–34] The other way is to deliver multiple shocks. The international electrical safety standard IEC-479–2 (IEC is the International Electrotechnical Commission) refers to shocks of 200 μC as "disagreeable" and 20,000 μC as "ventricular fibrillation likely" when delivered between the trunk and a limb.[35] Note that the values thought to be associated with VF is 200 times greater than the TASER ECD charge.[36] The X26 pulse has far less charge than the electric fence and has been found to satisfy both the IEC and USA Underwriter's Laboratories standards for such devices.

Even if it is impossible to induce VF with a single TASER ECD discharge, the possibility that VF might occur after multiple discharges must be analyzed. With a charge of 100 μC delivered 19 times per second, a TASER ECD delivers an average current of 1.9 mA. This value is at about two orders of magnitude below the current typically required to cause electrocution and thus that possibility appears to be essentially zero.[35]

2.3. Animal and modeling studies

With probes essentially across the heart VF induction is occasionally possible in very small swine (28–29 kg)[37,38] as they are more sensitive to electrical current than other mammals[39] probably due to the transmural penetration of the Purkinje fibers.[40] In the dog and human the Purkinje fibers are confined to an endocardial layer[41] while those of the pigs are transmural – penetrating the full myocardium. Thus, myocardial activation proceeds from the endocardium to the epicardium in dogs and humans while it proceeds from the epicardium to the endocardium in swine.[42] In other words, swine hearts are "wired" inside-out compared to human hearts. Swine are also exquisitely sensitive to high frequency electrical current.[43] Finally, the VF threshold is directly related to the body weight for both utility power and ECD waveforms.[39,44–46]

Modeling studies sponsored by the United Kingdome Home Office predict a safety margin of 240:1 for the X26 in adult humans.[47] Other modeling studies suggest that the currents reaching the heart are significantly below those required for stimulation.[13,19,48]

2.4. Human studies

The first published ECD human study included many volunteers with preexisting heart disease or diabetes (n = 66, age 40.3 ± 6.8 years).[49] Each volunteer was shot in the back with standard TASER probes and received the full 5-s application. Each had blood drawn before, immediately after, and at 16 and 24 h post-exposure. Troponin I, potassium, creatine kinase, lactate, and myoglobin were tested. A 12-lead EKG was recorded four times for each of 32 randomly chosen subjects (after each venipuncture). A blinded cardiologist read all 128 EKGs in random order. The TASER ECD did not affect cardiac or skeletal serum markers nor cause serial EKG changes.

Numerous other human studies have found no negative effects on the hearts of volunteers exposed to the TASER devices.[18,21,50–53] In one study continuous echocardiographic monitoring was used to monitor myocardial contractility and rhythm during ECD applications; it showed no cardiac capture and no decrease in cardiac output, even in exercised subjects.[16]

Finally, there is a case report describing a patient with an implanted ICD who received a sternal TASER ECD discharge. Interestingly, the ICD incorrectly interpreted the TASER current as VF but, as it takes more than 5 s for ICDs to detect an arrhythmia and charge, the "offending" rhyhm had disappeared by the time the ICD was ready to discharge.[54] Of note is that the patient suffered no complications from the entire episode.

3. Other mortality risks

3.1. Acidosis and rhabdomyolysis

Many arrest-related-deaths present with acidosis and this is to be expected from the effects of a prolonged struggle, hyperactivity, or drug intoxication.[55–60] Since the ECD causes moderate muscle contractions it is natural to consider the possibility that an ECD application might exacerbate this acidosis. Animal studies with ***unventilated*** anesthetized swine do report pH changes.[36,61,62] However, in ventilated swine and in human volunteers pH changes are not noted or are clinically insignificant.[49,52] Human studies out to 45-s applications have found no deleterious effects on breathing or pH.[64]

The moderate muscle contractions do not appear to be sufficient to present a risk of rhabdomyolysis as neither myoglobin nor creatine kinase levels increase significantly.[49,62]

3.2. Falls and traumatic brain injury

There are at least six cases of violently resisting subjects who experienced fatal traumatic head injuries from falls in which a TASER ECD may have contributed to the fall. This is unavoidable

as the design goal of the ECD is to cause a fall to stop a violent criminal suspect from advancing or struggling. These cases appear to represent the only scientifically linked deaths to ECD applications.

3.3. Summary of non-cardiac injuries and mortality

There are anecdotal reports of non-fatal complications secondary to TASER ECD application (superficial burns, globe penetration, and thoracic compression fracture), but such reports are rare.[65,66] While delivering a larger charge (100 μC) than earlier devices, the X26 delivers only 1.3 W of power (versus 7.4 W for the predecessor M26) and thus causes far less burning as there is less heating capability. Note that an infant night-light is typically 7 W.

Field usage studies suggest very high safety and significant reductions in injuries from the adoption of ECDs.[7,67–70] *It is significant that no human studies have ever suggested any direct mortality risk from these devices.*

4. Autopsy findings

4.1. Probe application

Central puncture wound. Depending on the postmortem interval a flat erythematous circular lesion (2–4 mm diameter) may be seen. Prolonged application times may result in the proximal surrounding tissue having a cauterized appearance. If the probes were not immediately pulled by police officers, there might be a small bleb or blister in the surrounding skin.

4.2. Drive stun application

Central abrasion with underlying tissue having a cauterized appearance. May have scattered abrasions as the electrode tip moves across the skin surface during application when the subject moves around vigorously during the application.

5. Conclusion

There is essentially zero risk of the induction of VF by shock delivered from TASER ECDs, and it matters not whether single or multiple shock are delivered. All of the available scientific evidence suggests that the output of the ECD is incapable of inducing VF in adult human beings. Human studies find no evidence of acidosis or rhabdomyolysis. ECDs have contributed to fatal fall injuries.

Conflict of interest statement

The author is a member of both the TASER International corporate and the scientific and medical advisory board.

References

1. International Electrotechnical Commission. Electromagnetic compatibility (EMC) - part 4-2: testing and measurement techniques – Electrostatic discharge immunity test. IEC.
2. Kroll M. Crafting the perfect shock. *IEEE Spect* 2007;**44**:27–30.
3. TASER_X26_Specifications. <http://www.taser.com/SiteCollectionDocuments/Controlled%20Documents/Spec%20Sheets/Law%20Enforcement/RD-SPEC-X26E-001_J.pdf>.
4. Roberts J. Medical effects of TASERs. *Emerg Med News* 2008;**30**:12–5.
5. Blain M. *Taser threat helps deter most suspects, national figures show Janes police review*. London, UK: Janes; 2008.
6. Vilke GM, Chan TC. Less lethal technology: medical issues. *Policing* 2007;**30**:341–57.
7. White M, Ready J. The TASER as a less lethal force alternative findings on use and effectiveness in a large metropolitan police agency. *Police Quart* 2007;**10**:170–91.
8. Sweeney J. Skeletal muscle response to electrical stimulation. In: Reilly J, editor. *Electrical stimulation and electropathology*. New York City: Cambridge University Press; 1992. [p. 285–327].
9. Reilly JP. *Applied bioelectricity: from electrical stimulation to electrical pathology*. New York: Springer; 1998. [vol. 33, p. 1–563].
10. Voorhees CR, Voorhees 3rd WD, Geddes LA, Bourland JD, Hinds M. The chronaxie for myocardium and motor nerve in the dog with chest-surface electrodes. *IEEE Trans Biomed Eng* 1992;**39**:624–8.
11. Yoon RS, DeMonte TP, Hasanov KF, Jorgenson DB, Joy ML. Measurement of thoracic current flow in pigs for the study of defibrillation and cardioversion. *IEEE Trans Biomed Eng* 2003;**50**:1167–73.
12. Sweeney J, Kroll M, Panescu D. Analysis of electrical activation of nerve and muscle by TASERs American Academy of Forensic Sciences. Seattle, WA: Publication Printers Corp, Denver, USA; 2006. p. 142–3.
13. Stratbucker RA, Kroll MW, McDaniel W, Panescu D. Cardiac current density distribution by electrical pulses from TASER devices. *Conf Proc IEEE Eng Med Biol Soc* 2006;**1**:6305–7.
14. Panescu D, Kroll MW, Efimov IR, Sweeney JD. Finite element modeling of electric field effects of TASER devices on nerve and muscle. *Conf Proc IEEE Eng Med Biol Soc* 2006;**1**:1277–9.
15. Panescu D, Webster JG, Stratbucker RA. Modeling current density distributions during transcutaneous cardiac pacing. *IEEE Trans Biomed Eng* 1994;**41**:549–55.
16. Ho JD, Dawes DM, Reardon RF, Lapine AL, Dolan BJ, Lundin EJ, et al. Echocardiographic evaluation of a TASER-X26 application in the ideal human cardiac axis. *Acad Emerg Med*.
17. Kroll MW, Calkins H, Luceri RM, Graham MA, Heegaard WG. Electronic control devices. *Cmaj* 2008;**179**:342–3.
18. Levine SD, Sloane CM, Chan TC, Dunford JV, Vilke GM. Cardiac monitoring of human subjects exposed to the taser. *J Emerg Med* 2007;**33**:113–7.
19. Panescu D, Kroll M, Stratbucker R. Finite element modeling of fibrillation risk from TASER conducted electrical weapons. *IEEE Trans Biomed Eng*.
20. Swerdlow C, Kroll M, Williams H, Biria M, Lakkireddy D, Tchou P. Presenting rhythm in sudden custodial deaths after use of TASER® electronic control device. *Heart Rhythm* 2008;**5**:S44.
21. Vilke GM, Sloane C, Levine S, Neuman T, Castillo E, Chan TC. Twelve-lead electrocardiogram monitoring of subjects before and after voluntary exposure to the Taser X26. *Am J Emerg Med* 2008;**26**:1–4.
22. Kroll M, Tchou P. Testing of implantable defibrillator functions at implantation. In: Ellenbogen K, Kay G, Lau C, Wilkoff B, editors. *Clinical cardiac pacing defibrillation and resynchronization therapy*. Philadelphia: W.B. Saunders Company; 2006. [p. 531–57].
23. Biegelmeier. Effect of current passing through the human body and the electrical impedance of the human body: a guide to IEC-Report 469. Berlin, ETZ: VDE-Verlag; 1987.
24. Biegelmeier G, Lee WR. New considerations on the threshold of ventricular fibrillation for a.c.shocks at 50–60 Hz. *IEE Proc* 1980;**127**(Pt. A):103–10.
25. Sharma AD, Fain E, O'Neill PG, Skadsen A, Damle R, Baker J, et al. Shock on *T* versus direct current voltage for induction of ventricular fibrillation: a randomized prospective comparison. *Pacing Clin Electrophysiol* 2004;**27**:89–94.
26. Weismuller P, Richter P, Binner L, Grossmann G, Hemmer W, Hoher M, Kochs V, Hombach V. Direct current application: easy induction of ventricular fibrillation for the determination of the defibrillation threshold in patients with implantable cardioverter defibrillators. *Pacing Clin Electrophysiol* 1992;**15**:1137–43.
27. Frame R, Brodman R, Furman S, Kim SG, Rot J, Ferrick K, et al. Clinical evaluation of the safety of repetitive intraoperative defibrillation threshold testing. *Pacing Clin Electrophysiol* 1992;**15**:870–7.
28. Stratton SJ, Rogers C, Brickett K, Gruzinski G. Factors associated with sudden death of individuals requiring restraint for excited delirium. *Am J Emerg Med* 2001;**19**:187–91.
29. Park KS, Korn CS, Henderson SO. Agitated delirium and sudden death: two case reports. *Prehosp Emerg Care* 2001;**5**:214–6.
30. DiMaio T, VJM D. *Excited delirium syndrome cause of death and prevention*. Boca Raton: Taylor & Francis; 2006.
31. Schipke JD, Heusch G, Sanii AP, Gams E, Winter J. Static filling pressure in patients during induced ventricular fibrillation. *Am J Physiol Heart Circ Physiol* 2003;**285**:H2510–5.
32. Green R, Rafferty E, Gregory J. Danger levels of short (1 ms–15 ms) electrical shocks from 50 Hz power supply. In: Bridges J, Ford G, Sherman I, Vainberg M, editors. *Electrical shock safety criteria*. New York: Pergamon; 1985. [p. 259–72].
33. Peleska B. Cardiac arrhythmias following condenser discharges and their dependence upon strength of current and phase of cardiac cycle. *Circulation Research* 1963;**13**:21–32.
34. Wiggers CJ, Wegria R. Ventricular fibrillation due to single, localized induction and condenser shocks applied during the vulnerable phase of ventricular systole. *Am J Physiol J1 – AJP* 1940;**128**:500–5.
35. International Electrotechnical Commission. Effects of Current on Human Beings and Livestock, CEI/IEC 479-2-Effects of currents passing through the human body. 2nd ed. Geneva, Switzerland: IEC; 1987.
36. Nimunkar AJ, Webster JG. Safety of pulsed electric devices. *Physiol Meas* 2009;**30**:101–14.
37. Dennis AJ, Valentino DJ, Walter RJ, Nagy KK, Winners J, Bokhari F, et al. Acute effects of TASER X26 discharges in a swine model. *J Trauma* 2007;**63**:581–90.
38. Walter RJ, Dennis AJ, Valentino DJ, Margeta B, Nagy KK, Bokhari F, et al. TASER X26 discharges in swine produce potentially fatal ventricular arrhythmias. *Acad Emerg Med* 2008;**15**:66–73.

39. Ferris LP, King BG, Spence PW, Williams HB. Effect of electric shock on the heart. *Electr Eng* 1936;**55**:498–515.
40. Howe BB, Fehn PA, Pensinger RR. Comparative anatomical studies of the coronary arteries of canine and porcine hearts. I. free ventricular walls. *Acta Anat (Basel)* 1968;**71**:13–21.
41. Schnabel PA, Richter J, Schmiedl A, Bach F, Bartels U, Ramsauer B, et al. Patterns of structural deterioration due to ischemia in Purkinje fibers and different layers of the working myocardium. *Thorac Cardiovasc Surg* 1991;**39**:174–82.
42. Allison JS, Qin H, Dosdall DJ, Huang J, Newton JC, Allred JD, et al. The transmural activation sequence in porcine and canine left ventricle is markedly different during long-duration ventricular fibrillation. *J Cardiovasc Electrophysiol* 2007;**18**:1306–12.
43. Pak HN, Kim YH, Lim HE, Chou CC, Miyauchi Y, Fang YH, et al. Role of the posterior papillary muscle and Purkinje potentials in the mechanism of ventricular fibrillation in open chest dogs and swine: effects of catheter ablation. *J Cardiovasc Electrophysiol* 2006;**17**:777–83.
44. Dalziel CF, Lee WR. Reevaluation of lethal electric currents. *IEEE Trans Ind Gen Appl* 1968;**IGA-4**:467–76.
45. Geddes LA, Cabler P, Moore AG, Rosborough J, Tacker WA. Threshold 60-Hz current required for ventricular fibrillation in subjects of various body weights. *IEEE Trans Biomed Eng* 1973;**20**:465–8.
46. McDaniel WC, Stratbucker RA, Nerheim M, Brewer JE. Cardiac safety of neuromuscular incapacitating defensive devices. *Pacing Clin Electrophysiol* 2005;**28**(Suppl. 1):S284–7.
47. Holden SJ, Sheridan RD, Coffey TJ, Scaramuzza RA, Diamantopoulos P. Electromagnetic modeling of current flow in the heart from TASER devices and the risk of cardiac dysrhythmias. *Phys Med Biol* 2007;**52**:7193–209.
48. Panescu D, Kroll M, Efimov I, Sweeney J. Finite element modeling of electric field effects of taser devices on nerve and muscle IEEE Eng Med Biol. New York City, NY; 2006;1:1277–9.
49. Ho JD, Miner JR, Lakireddy DR, Bultman LL, Heegaard WG. Cardiovascular and physiologic effects of conducted electrical weapon discharge in resting adults. *Acad Emerg Med* 2006;**13**:589–95.
50. Chan T, Sloane C, Neuman T, Levine S, Castillo E, Vilke G, et al. The impact of the taser weapon on respiratory and ventilatory function in human subjects. *Acad Emerg Med* 2007;**14**:191–2.
51. Dawes DM, Ho JD, Johnson MA, Lundin E, Janchar TA, Miner JR. 15-S conducted electrical weapon exposure does not cause core temperature elevation in non-environmentally stressed resting adults. *Forensic Sci Int* 2008;**176**:253–7.
52. Ho JD, Dawes DM, Bultman LL, Moscati RM, Janchar J, Miner JR. Prolonged TASER® use on exhausted humans does not worsen markers of acidosis. Am J Emerg Med; 2009, in press.
53. Ho JD, Dawes DM, Bultman LL, Thacker JL, Skinner LD, Bahr JM, et al. Respiratory effect of prolonged electrical weapon application on human volunteers. *Acad Emerg Med* 2007;**14**:197–201.
54. Haegeli LM, Sterns LD, Adam DC, Leather RA. Effect of a Taser shot to the chest of a patient with an implantable defibrillator. *Heart Rhythm* 2006;**3**:339–41.
55. Brice JH, Pirrallo RG, Racht E, Zachariah BS, Krohmer J. Management of the violent patient. *Prehosp Emerg Care* 2003;**7**:48–55.
56. Allam S, Noble JS. Cocaine-excited delirium and severe acidosis. *Anaesthesia* 2001;**56**:385–6.
57. Hick JL, Smith SW, Lynch MT. Metabolic acidosis in restraint-associated cardiac arrest: a case series. *Acad Emerg Med* 1999;**6**:239–43.
58. Chan TC, Neuman T, Vilke GM, Clausen J, Clark RF. Metabolic acidosis in restraint-associated cardiac arrest. *Acad Emerg Med* 1999;**6**:1075–6.. author reply, p. 1076-7.
59. Bethke RA, Gratton M, Watson WA. Severe hyperlactemia and metabolic acidosis following cocaine use and exertion. *Am J Emerg Med* 1990;**8**:369–70.
60. Bozzuto TM. Severe metabolic acidosis secondary to exertional hyperlactemia. *Am J Emerg Med* 1988;**6**:134–6.
61. Jauchem JR, Cook MC, Beason CW. Blood factors of Sus scrofa following a series of three TASER(R) electronic control device exposures. *Forensic Sci Int* 2008;**175**(2–3):166–70.
62. Jauchem JR, Sherry CJ, Fines DA, Cook MC. Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of Sus scrofa following repeated TASER exposures. *Forensic Sci Int* 2006;**161**:20–30.
64. Ho J, Dawes D. The effect of the extended range electronic projectile (XREP) on breathing. In: Australian college of emergency medicine winter symposium. Newcastle, NSW; 2008.
65. Winslow JE, Bozeman WP, Fortner MC, Alson RL. Thoracic compression fractures as a result of shock from a conducted energy weapon: a case report. *Ann Emerg Med* 2007;**50**:584–6.
66. Rehman TU, Yonas H, Marinaro J. Intracranial penetration of a TASER dart. *Am J Emerg Med* 2007;**25**. [p. 733 e3–4].
67. Bozeman W, Winslow J, Hauda W, Graham D, Martin B, Heck J. Injury profile of TASER® electrical conducted energy weapons (CEWs). *Ann Emergency Med* 2007;**50**:S65.
68. Mesloh C, Wolf R, Henych M, Thompson F. Less lethal weapons for law enforcement: a performance-based analysis. *Law Enforcement Executive Forum* 2008;**8**:133–49.
69. Smith R. TASER® non-lethal weapons: safety data and field results american academy of forensic sciences. Seattle: WA; 2006. p. 137–9.
70. Brewer J, Kroll M. Field statistics overview. In: Kroll MW, Ho JD, editors. *TASER conducted electrical weapons: physiology, pathology, and law*. New York City: Springer-Kluwer; 2009.